

Numbers of patients attending accident and emergency department on basis of a 999 call, with outcomes

Year	No of patients attending	Outcome			
		No (%) admitted	No (%) followed up	No (%) discharged	No (%) who died
1993	10 038	4407 (43.9)	890 (8.8)	4436 (44.1)	86 (0.85)
1994	11 212	4954 (44.2)	983 (7.6)	4937 (44.0)	103 (0.91)
1995	11 985	5179 (43.2)	941 (10)	5457 (45.5)	107 (0.89)
1996	12 896	5654 (43.8)	925 (7)	5887 (45.6)	120 (0.93)

come measures of admission, follow up, and discharge, which have been previously validated.<sup>5</sup> Patients who dialled 999 but were not transported to hospital were excluded as were patients whose admission had been arranged by their general practitioner.

The table shows patient numbers and outcomes. Total outcomes were slightly less than 100% (97.82%) because of information that had been insufficiently coded. The average annual increase in patients attending the accident and emergency department on the basis of a 999 call was 8.9%, which relates closely to an 8.6% increase in the numbers admitted. The difference in the percentage of patients admitted is greatest in the years 1995 and 1996 but comparison of the proportions admitted is not statistically significant ( $P=0.171$ ). There had therefore been no significant change in the proportion of people attending the accident and emergency department on the basis of a 999 call in the past four years.

### Comment

The annual increase in emergency admissions is a cause for concern for both healthcare workers and purchasers. To date, none of the reasons postulated for this increase can explain the size of the rise. As the

increase in numbers is far in excess of rises in bed capacity there is a commensurate increase in pressures on elective activity.

If the rise in 999 calls indicated an increase in the number of inappropriate calls the percentage of patients admitted based on those calls would fall. The numbers of patients admitted to accident and emergency departments have, however, risen in parallel with those admitted on the basis of a 999 call.

Contributors: The original idea for this study came from HG, who also collected the data for attendances, and reviewed and edited the paper. CM collected the ambulance data, undertook the literature search, analysed the statistics, and wrote and redrafted the paper; CM will act as guarantor of the study.

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Conflict of interest: None.

- 1 National Association of Health Authorities and Trusts. *Emergency admissions: the management challenge*. Birmingham: NAHAT, 1996.
- 2 Ley R. By inspection—emergency healthcare at the cross-roads: from supply-led demand to demand-led supply. Ipswich: Suffolk Health Authority, 1995.
- 3 Edwards E, Werneke U. In the fast lane. *Health Serv J* 1994;8:30-2.
- 4 Brown E, Sindelar J. The emergent problem of ambulance misuse. *Ann Emerg Med* 1993;22(4):646-50.
- 5 Anthony P, Blayden B, Bray P. *Casemix measures in accident and emergency medicine: an introductory review*. Winchester: National Casemix Office, 1995.

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## Raynaud's phenomenon after sympathetic denervation in patients with primary autonomic failure: questionnaire survey

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In Raynaud's phenomenon, cold or emotional stimuli cause digital vasospasm in the fingers and toes, leading to the classic colour triad sequence of pallor, cyanosis, and redness. Secondary Raynaud's phenomenon occurs in association with underlying disease, including neurological disorders.<sup>1</sup> We describe Raynaud's phenomenon in patients with sympathetic denervation due to primary chronic autonomic failure.

### Subjects, methods, and results

Seventy three patients with primary chronic autonomic failure were assessed for Raynaud's phenomenon. They all had orthostatic hypotension due to sympathetic failure, which was confirmed on detailed investigation.<sup>2</sup> Thirty two patients had pure autonomic failure and no other neurological features and 41 had multiple system atrophy (the Shy-Drager syndrome) with parkinsonian or cerebellar features, or both. None

had clinical features or disease usually associated with Raynaud's phenomenon. All the patients consented to answer a questionnaire, approved by the ethics committee of St Mary's Hospital, London.

Patients were considered to be positive for Raynaud's phenomenon if they had a definite sensitivity to cold. Overall, 51 patients had Raynaud's phenomenon, all showing the classic colour sequence triad in their hands (table); four patients were unsure of the sequence of colour change (one had pure autonomic failure and three multiple system atrophy). Over half of the patients reported a worsening of the condition in their hands and feet during cold weather. Under half of the patients reported that rubbing and warmth provided relief in their hands and feet within 15 minutes. Overall, 34 patients had Raynaud's phenomenon before being treated for orthostatic hypotension—all were prescribed fludrocortisone and the majority were taking the sympathomimetic vasoconstrictors

Raynaud's phenomenon in two groups of patients with primary chronic autonomic failure. Values are numbers (percentages) of patients unless stated otherwise

	Pure autonomic failure (n=32)	Multiple system atrophy (n=41)
Mean age (range) (years)	67 (47-82)	62 (45-79)
Sex:		
Male	16 (50)	27 (66)
Female	16 (50)	14 (34)
Mean duration of illness (range) (years)	11 (3.5-20)	5 (1-15)
<b>Raynaud's phenomenon</b>		
In hands	20 (63)	31 (76)
In feet	13 (41)	22 (54)
Worse in cold weather:		
In hands	11/20 (55)	20/31 (65)
In feet	9/13 (69)	17/22 (77)
Relieved by rubbing and warmth:		
In hands:		
<5 minutes	1/20 (5)	2/31 (6)
5 to 15 minutes	8/20 (40)	11/31 (35)
In feet		
<5 minutes	1/13 (8)	0/22 (0)
5 to 15 minutes	5/13 (38)	9/22 (41)
Present before drug treatment:		
In hands	13/20 (65)	21/31 (68)
In feet	9/13 (69)	14/22 (64)
Worse after drug treatment:		
In hands	2/20 (10)	4/31 (13)
In feet	2/13 (15)	2/22 (9)

ephedrine or midodrine. In most patients drug treatment did not worsen the condition. Patients reported similar findings in their hands and feet.

## Comment

This study confirms that Raynaud's phenomenon and cold sensitivity in the hands and feet commonly occur in patients with sympathetic denervation due to primary chronic autonomic failure. We did not assess the effect of emotional stimuli on Raynaud's phenomenon in our patients. Most patients described the classic symptoms of the phenomenon in their hands and feet. Published data indicate that the prevalence of Raynaud's phenomenon varies from 0.5% to 20% according to population, age, and sex.<sup>1</sup> The incidence was considerably higher in our patients with autonomic failure. None of them had an underlying disorder known to be associated with Raynaud's phenomenon, and most were followed up for over 1 year. None of our patients presented with Raynaud's phenom-

enon; we did not inquire at what stage in their illness it occurred. In most of them drug treatment did not seem to induce or worsen it.

Cold sensitivity in Raynaud's phenomenon may be linked to sympathetic activation.<sup>3</sup> Surgical sympathectomy usually is not effective in treating Raynaud's phenomenon, probably because adrenoceptors in limb vessels will still interact after such surgery with circulating catecholamines, including those released from the adrenal medulla. In primary chronic autonomic failure the function of the adrenal medulla usually is impaired but there is pressor supersensitivity to noradrenaline<sup>4</sup>; whether residual sympathetic activity (however minimal) along with supersensitivity contributes to cold sensitivity and Raynaud's phenomenon is unclear. There was no important difference in the frequency of Raynaud's phenomenon in patients with pure autonomic failure, who have peripheral lesions and low plasma noradrenaline concentrations, when compared with patients with multiple system atrophy, who have central autonomic lesions and basal plasma noradrenaline concentrations usually within the normal range. Calcitonin gene related peptide and nitric oxide seem deficient in primary Raynaud's phenomenon<sup>5</sup>; it is not known whether such abnormalities occur in primary chronic autonomic failure.

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- 1 Black CM. Update on Raynaud's phenomenon. *Br J Hosp Med* 1994;52:555-7.
- 2 Mathias CJ. Disorders of the autonomic nervous system. In: Bradley WG, Daroff RB, Fenichel GM, Marsden CD, eds. *Neurology in clinical practice*. Vol II. *The neurological disorders*. 2nd ed. Boston, MA: Butterworth-Heinemann, 1996;8:1953-81.
- 3 Jamieson GG, Ludbrook J, Wilson A. Cold hypersensitivity in Raynaud's phenomenon. *Circulation* 1971;44:254-64.
- 4 Polinsky RJ. Neuropharmacological investigation of autonomic failure. In: Bannister R, Mathias CJ eds. *Autonomic failure—a textbook of clinical disorders of the autonomic nervous system*. 3rd ed. Oxford: Oxford University Press, 1992:334-58.
- 5 Dowd PM, Goldsmith PC, Bull HA, Bunnstock G, Roreman JC, Marshall I. Raynaud's phenomenon. *Lancet* 1995;346:283-9.

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## *Fifty years ago* The new NHS

SIR,—If the majority of us G.P.s are opposed to the National Health Service Act and if we refuse to join the Service, surely the Act cannot be worked and an amendment will be forced. Assuming a majority against the Act, our weakness lies in our lack of team spirit. We are not sure of the strength of will of our colleagues in other parts of the country. We fear that a trickle into the Service may increase to a rapid flow, followed in the end by a landslide.

I suggest that as a part of the questionnaire the profession be asked: "If you are opposed to the Service as it stands, and if X%

(say 60) are of the same opinion, will you bind yourself to refuse to join the Service until acceptable terms are agreed by the Negotiating Committee?" If 60% bound themselves thus, our minds would be greatly relieved, and the arm of the Negotiating Committee would be immensely strengthened.—I am, etc,  
G. H. Gibbens, Sidmouth, Devon.

(*Letter*, 17 January 1948, p 116. See also editorial by Gordon Macpherson, 3 January 1998, p 6.)